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Indoor Air Pollution and Pulmonary Function Growth in Preadolescent Children

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16. ABSTRACT

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INDOOR AIR POLLUTION AND PULMONARY FUNCTION GROWTH IN PREADOLESCENT CHILDREN

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forced expiratory volume; forced vital capacity; longitudinal studies; nitrogen dioxide; smoking, passive; tobacco smoke pollution

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Abbreviations: FEV_1, forced expiratory volume in one second; FVC, forced vital capacity.

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Although outdoor air pollution has long been recognized as a potential health hazard, the contaminants of indoor air from either the passive diffusion of outdoor air to the indoors or from indoor sources have only recently received similar attention. A recent National Academy of Science report (1) concluded that the principal population exposure to several pollutants may be associated with activities inside the home. In particular, "for many people the main or sole exposure to numerous gaseous and particulate compounds results from passive exposure to tobacco smoke," (1, page V-2) and "unvented gas cooking is probably responsible for a large portion of nitrogen dioxide exposures in our population" (1, page ES-8).

Several epidemiologic studies have reported higher frequencies of respiratory illnesses or reduced pulmonary function levels among children exposed to sidestream cigarette smoke (2–7). Although some investigators have not found such associations (8–11), the bulk of the evidence strongly supports a positive relationship. The evidence regarding the health effects of gas stove emissions has been less consistent. Melia and coworkers (12, 13) reported increased illness and symptom rates but no reduction in pulmonary function level among children exposed to gas stove emissions, while Speizer and colleagues (7, 14) found lower pulmonary function levels but no increases in illness or symptom frequencies. Keller et al. (15) reported no associations and Hasselblad et al. (4) found reduced pulmonary function levels only among older girls.

Since each of these studies relied on cross-sectional data, little is known about the association between exposure to indoor air pollutants and growth rate of pulmonary function. A recent report, however, has suggested that pulmonary function growth rates may be affected by exposure to sidestream cigarette smoke (16). The Harvard Study of Air Pollution and Health, an ongoing longitudinal investigation of the health effects of air pollutants, both indoor and outdoor, provides an opportunity for further investigation of this question. This report describes the association between the rates of change of two measures of pulmonary function—forced expiratory volume in one second and forced vital capacity—and exposure to sidestream cigarette smoke or gas stove emissions among a sample of 7,834 children receiving spirometric examinations on two or more occasions between their sixth and tenth birthdays. Estimated pulmonary function levels at eight years of age are also investigated for associations with pollutant exposure.

Materials and Methods

The study enrolled 12,252 children in six geographic areas of the eastern and midwestern United States (Watertown, MA; Kingston and Harriman, TN; Steubenville and Mingo Junction, OH; a geographically defined section of St. Louis, MO; Portage, WI and several surrounding communities; and Topeka, KS) between 1974 and 1981. At the initial and subsequent annual examinations, children were seen in their schools for determination of height and weight and for a spirometric examination. At each visit, children took home a standardized questionnaire to be completed by a parent or guardian. The questionnaire requested information about parental smoking habits, type of cooking fuel used in the home, parental education, and respiratory illness and symptom histories for the participating child and the parents. Each child in grade four or higher was asked privately about personal smoking habits. Additional information regarding the study design is provided elsewhere (17).

Standing height and weight were measured in stocking feet and the children performed forced expiratory maneuvers on a water-filled recording spirometer (Survey Spirometer, Warren E. Collins, Braintree, MA) while sitting with free mobility and without a noseclip. Each child performed at least five forced expirations but not more than eight. Forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1) were measured for each blow judged acceptable by the examiner. The mean of

the best three efforts was calculated after correction for body temperature and water saturation. A more detailed description of the measurement procedures is given elsewhere (18).

The analyses presented were restricted to white children with at least two acceptable pulmonary function measurements between their sixth and tenth birthdays. We excluded five-year-olds because of the poorer cooperation and greater learning effect seen in this age group (18). The tenth birthday was selected as the upper limit to avoid the adolescent growth spurt for most children. Dickman et al. (19) found that the pulmonary function growth spurt begins when the child is about 154 cm tall, which fewer than 3 per cent of children reach by age 10 (20). Detels et al. (21) found that peak growth velocity for FEV_1 occurred about one year after peak growth velocity for height, which occurred at age 10 for girls and at age 12 for boys.

For this report, an examination was excluded if the child reported smoking at least one cigarette per week at the time of the examination. The data for each child were reviewed longitudinally, as described in the next section, to identify atypical values.

Statistical methods

For each spirometric examination, we produced a predicted FEV_1 and FVC based on the child's height, weight, sex, and age. Previous work had shown that analyses of children's pulmonary function measurements should utilize the natural logarithmic transformation (18). For this analysis, predicted values for the logarithms of FEV_1 and FVC were calculated using the model developed in that work, but with regression coefficients re-estimated from the data set analyzed for this report. The model included the logarithms of height, weight, and age plus an indicator variable for sex and a sex-specific height term. An FEV_1 residual, defined as the difference between the logarithms of the observed and predicted FEV_1 values, was then calculated for each examination. An FVC residual was calculated similarly.

To identify children with inconsistent observations, standard deviations of these FEV_1 residuals, and separately of the FVC residuals, were computed for each child. Sixty-six children had strikingly large standard deviations for either FEV_1 or FVC (more than 5 standard deviations from the sample mean). The serial values for each of these children were reviewed. In each instance, a clearly erroneous value was found and the FEV_1 and FVC for that examination were deleted from that child's record.

Analysis of the FEV_1 or FVC residuals was based on a two-step growth curve method (22). In the first step of the analysis, each child's series of pulmonary function residuals was regressed on the ages (centered at eight years) at the successive examinations. The coefficients of the fitted regression line provided estimates of the child's pulmonary function *growth rate* and *level* at age eight. The antilogarithm of this growth rate times 100 expresses each child's rate of change of pulmonary function as a percentage of the population mean annual growth rate. The antilogarithm of the level times 100 is the child's per cent of predicted pulmonary function at age eight. The expression of pulmonary function level at an age common to all children was required for comparability in subsequent analysis of risk factors. For clarity, we report the level and growth rate as differences from 100 per cent. Thus a child whose observed pulmonary function values were all equal to the predicted values would have a level of 0 per cent and growth rate of 0 per cent per year.

Figure 1 illustrates this method of summarizing the FEV_1 values for an individual child, a girl who had four examinations. Her logarithm (FEV_1) values are plotted against the four ages; also shown are the predicted logarithm (FEV_1) values. The differences between each pair of points are the four FEV_1 residuals. The regression line fitted to these four residuals yields the estimated FEV_1 growth rate and level for this child.

The second step of the growth curve method used the coefficients of these fitted

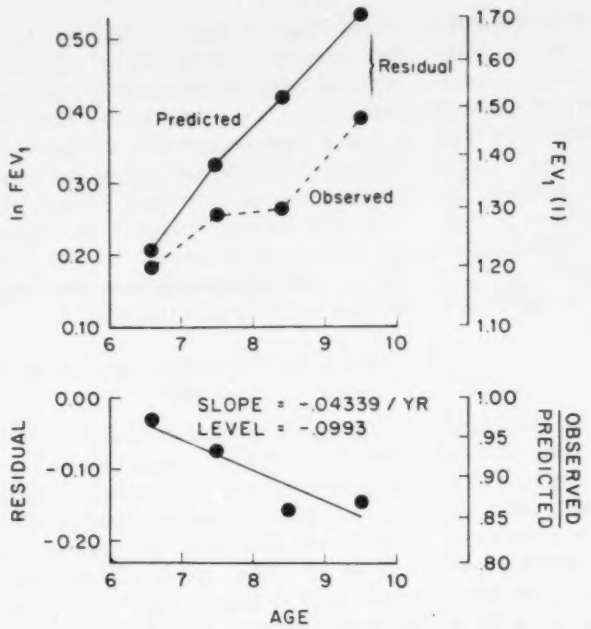

FIGURE 1. Calculation of growth rate and level of $\ln(FEV_1)$ for an individual child. The residuals in the upper panel. i.e., the difference between observed and predicted $\ln(FEV_1)$, were regressed on age in the lower panel.

regression lines as the dependent variables in a further regression analysis. Specifically, the estimated levels and growth rates for FEV_1 and FVC (thus, four dependent variables) were analyzed separately by weighted least squares regression. Each weighting variable was the inverse of the estimated variance of the individual level or slope, defined as the sum of the between-subject and within-subject variances. The between-subject variance was common to all children, but the within-subject variance, and therefore the weight, depended on the number of examinations and the ages at examination. In particular, children with only two observations received the lowest weights. The variance components were estimated by an iterative procedure based on the methodology of Hui and Berger (23) and Fay and Herriot (24). These weights also were used to calculate weighted means of FEV_1 and FVC level and growth rate for the full sample and for subgroups defined by values of demographic variables and risk factors. All weighted means and regression coefficients were exponentiated and expressed in per cent.

The independent variables of primary interest included parental smoking habits and type of cooking stove. Maternal and paternal smoking *status* was defined by classifying each parent as a smoker if they were reported to be a current smoker at any examination and a nonsmoker otherwise. Maternal and paternal smoking *level* was defined as the average over examinations of number of cigarettes smoked per day. Stove type was defined as the proportion of examinations with a positive response for gas stove.

Four different models for effects of parental smoking were considered (table 4). Model 1 included both maternal and paternal smoking status, Model 2 included only maternal smoking status, and Model 3 included the number of parents who smoked. Model 4 included maternal smoking level to investigate the existence of an exposure-response relationship. Each analysis also

included indicator variables identifying the geographic area of residence and a three-level variable for socioeconomic status based on the average number of years of education of the child's parents (less than nine, nine to 12, more than 12). Other variables considered in the analysis included a reported history (yes/no) of physician-diagnosed respiratory illness before two years of age and a history (yes/no) of bronchitis prior to the initial examination.

Results

Sample

Between the 1974–1975 and 1980–1981 school years, 12,252 children less than 10 years of age were seen at least once. Of these, 11,140 were white and 1,112 were from other racial groups. This analysis is restricted to the 7,867 white children who had at least two acceptable pulmonary function examinations during that period. Fourteen measurements were eliminated because the child reported smoking at least one cigarette per week at the time of the examination. Ninety-nine additional measurements were excluded because they were inconsistent with the other serial measurements from the same child; this included the complete elimination of 33 children with two inconsistent observations. The remaining 22,901 observations from 7,834 children represented 88.4 per cent of all observations collected for white children between six and 10 years of age. When examinations are grouped by half-years of age, the mean values of the unadjusted FEV_1, height, and weight are slightly higher for boys than for girls (table 1).

Demographic variables and respiratory health

The mean (deviation from predicted) FEV_1 level at eight years of age for the 7,834 children was −0.02 per cent (table 2) and the mean (deviation from predicted) growth rate was +0.03 per cent per year. Neither of these values differed significantly from 0. Mean FVC level was −0.13 per cent and mean growth rate was +0.15 per cent per year. The growth rate for FVC was significantly different from 0 ($p < 0.01$). Mean values for boys and girls were not significantly different for any measure (table 2).

Earlier reports from this study (18) in-

TABLE 1

Mean FEV_1, FVC, height, and weight by age and sex for 7,834 children examined 2–5 times between their 6th and 10th birthdays

Age group (years)	No.	FEV_1 (liters)	FVC	Height (meters)	Weight (kg)
			Boys		
6–	456	1.294	1.471	1.181	22.75
6.5–	982	1.356	1.544	1.204	24.05
7–	1,341	1.440	1.656	1.236	25.38
7.5–	1,655	1.519	1.749	1.260	26.67
8–	1,865	1.617	1.873	1.291	28.40
8.5–	1,849	1.692	1.969	1.318	30.10
9–	1,739	1.787	2.083	1.346	31.80
9.5–<10	1,660	1.866	2.181	1.373	33.82
			Girls		
6–	480	1.210	1.352	1.174	22.14
6.5–	1,002	1.276	1.429	1.197	23.43
7–	1,299	1.373	1.539	1.229	25.06
7.5–	1,700	1.425	1.605	1.253	26.29
8–	1,745	1.519	1.714	1.285	28.15
8.5–	1,827	1.593	1.807	1.311	29.71
9–	1,689	1.690	1.917	1.341	31.73
9.5–<10	1,612	1.768	2.010	1.369	33.63

TABLE 2

Weighted means (standard errors) of pulmonary function levels and growth rates (deviations from predicted) of 7,834 children, by sex, number of examinations, city of residence, parental education, and illness history

Variable	No.	FEV_1		FVC	
		Level (%)	Growth rate (%/year)	Level (%)	Growth rate (%/year)
Total	7,834	−0.02 (0.12)	+0.03 (0.05)	−0.13 (0.12)	+0.15 (0.05)
Sex					
Boys	3,989	−0.03 (0.27)	+0.03 (0.07)	−0.04 (0.17)	+0.14 (0.07)
Girls	3,845	−0.08 (0.17)	+0.03 (0.07)	−0.21 (0.17)	+0.17 (0.07)
No. of examinations					
2	2,677	−0.53 (0.24)	+0.34 (0.14)	−0.72 (0.22)	+0.84 (0.12)
3	3,099	+0.26 (0.19)	+0.10 (0.08)	+0.09 (0.18)	+0.24 (0.07)
4	2,040	+0.05 (0.22)	−0.11 (0.07)	+0.16 (0.22)	−0.18 (0.07)
5	18	−2.15 (2.34)	−0.09 (0.66)	−1.17 (2.33)	−1.00 (0.71)
3+	5,157	+0.17 (0.14)	+0.02 (0.06)	+0.11 (0.14)	+0.07 (0.05)
City					
Portage, WI	1,250	+1.92 (0.31)	−0.41 (0.12)	+2.19 (0.30)	−0.61 (0.12)
Topeka, KS	1,216	−0.97 (0.32)	+1.06 (0.14)	−1.06 (0.31)	+1.71 (0.13)
Watertown, MA	1,334	−1.06 (0.29)	+0.15 (0.11)	−2.25 (0.28)	+0.18 (0.11)
Kingston, TN	993	−0.64 (0.35)	−0.25 (0.14)	+0.11 (0.34)	−0.26 (0.14)
St. Louis, MO	1,546	−0.07 (0.28)	−0.01 (0.11)	−0.19 (0.27)	+0.23 (0.11)
Steubenville, OH	1,495	+0.50 (0.28)	−0.19 (0.11)	+0.51 (0.27)	−0.08 (0.11)
Parental education					
< High school	980	−0.68 (0.35)	−0.15 (0.15)	−0.37 (0.34)	+0.00 (0.14)
High school	3,948	−0.06 (0.17)	−0.02 (0.07)	−0.21 (0.17)	+0.11 (0.07)
> High school	2,665	+0.31 (0.21)	+0.18 (0.08)	+0.19 (0.20)	+0.30 (0.08)
Missing	241				
Physician-diagnosed respiratory illness at <2 years old					
No	6,600	+0.05 (0.13)	−0.02 (0.05)	−0.00 (0.13)	+0.19 (0.05)
Yes	214	−1.83 (0.75)	−0.01 (0.30)	+0.35 (0.72)	−0.13 (0.29)
Missing	1,020				
History of bronchitis					
No	6,640	+0.16 (0.13)	+0.02 (0.05)	−0.15 (0.13)	+0.17 (0.05)
Yes	1,143	−1.03 (0.32)	+0.03 (0.13)	+0.11 (0.31)	+0.07 (0.12)
Missing	51				

dicated that pulmonary function measurements at the initial examination were often lower than the predicted values, presumably due to a learning effect which occurs subsequent to this initial examination. Theoretically, this effect of first examination should be most noticeable among children with only two measurements, whose levels will be negatively biased and growth rates will be positively biased. In fact, children with only two measurements (table 2) had a statistically significant negative mean level of FEV_1 (−0.53 per cent) and a statistically significant positive mean growth rate (+0.34 per cent per year). For children with three or more visits, the mean FEV_1 level (+0.17 per cent) and mean relative growth rate (+0.02 per cent per year) were not significantly different from 0. Similar results were found for FVC (table 2).

Parental education, a measure of socioeconomic status, was associated with both level and growth rate. Children whose parents were least educated had the lowest mean level and the lowest mean growth rate for both FEV_1 and FVC (table 2). Both level and growth rate increased monotonically with level of parental education. All regression analyses to test for effects of parental smoking or gas stoves were therefore adjusted for parental education.

Two indicators of early respiratory illness, reported physician-diagnosed respiratory illness before two years of age and reported history of bronchitis, were associated with significantly lower mean FEV_1 levels ($p < 0.02$ and $p < 0.001$, respectively), but not with growth rates (table 2). No associations were found between either measure of early respiratory illness and FVC level or growth rate. These two indicators of early respiratory illness were not considered in the subsequent analyses.

Parental smoking

The mean values of FEV_1 level and growth rate by parental smoking status suggest an independent effect of each parent's smoking on both outcome variables (table 3). Children whose parents did not smoke had both the highest level and highest growth rate. A regression analysis including maternal and paternal smoking status, city of residence, and parental education gave an estimated effect of maternal smoking on FEV_1 level of −0.61 per cent ($p = 0.03$) and for paternal smoking of −0.41 per cent ($p = 0.14$) (table 4). For FEV_1 growth rate, the estimated effect of maternal smoking was −0.09 per cent per year ($p = 0.43$) and the estimated effect of paternal smoking was −0.02 per cent per year ($p = 0.87$). Since paternal and maternal smoking behavior were positively correlated, the analysis was repeated with only maternal smoking included in the model, yielding an estimated effect of −0.80 per cent ($p = 0.002$) for FEV_1 level, and −0.10 per cent per year ($p = 0.32$) for FEV_1 growth rate. A weighted regression analysis including the number of parents who smoked as the independent variable gave estimated effects of −0.47 per cent ($p = 0.003$) per smoker for FEV_1 level and −0.09 per cent per year ($p = 0.19$) per smoker for growth rate.

To assess the evidence for an exposure-response relationship, parental smoking level was then defined as the number of cigarettes smoked by each parent. A regres-

TABLE 3

Weighted means (standard errors) of pulmonary function levels and growth rates (deviations from predicted), of 7,834 children by parental smoking habits and stove type

Variable	No.	FEV_1		FVC	
		Level (%)	Growth rate (%/year)	Level (%)	Growth rate (%/year)
Parental smoking					
Neither	2,042	+0.65 (0.24)	+0.15 (0.10)	−0.02 (0.23)	+0.21 (0.09)
Father only	1,877	+0.26 (0.25)	−0.04 (0.10)	−0.36 (0.24)	+0.16 (0.10)
Mother only	738	+0.02 (0.40)	−0.08 (0.15)	−0.06 (0.39)	−0.06 (0.15)
Both	2,484	−0.54 (0.22)	+0.01 (0.09)	+0.08 (0.21)	+0.15 (0.08)
Missing	693				
Maternal smoking (cigarettes/day)					
0	4,208	+0.45 (0.17)	+0.08 (0.07)	−0.18 (0.16)	+0.21 (0.06)
1–5	431	+0.41 (0.52)	+0.36 (0.20)	−0.30 (0.50)	+0.49 (0.19)
6–15	916	−0.14 (0.36)	−0.13 (0.14)	−0.20 (0.35)	+0.05 (0.14)
16–25	1,446	−0.79 (0.29)	+0.02 (0.11)	+0.19 (0.28)	−0.00 (0.11)
26–35	534	−1.86 (0.47)	−0.23 (0.19)	−0.82 (0.46)	+0.03 (0.18)
36–45	202	−0.55 (0.77)	−0.32 (0.31)	+0.72 (0.74)	+0.08 (0.30)
46+	53	+1.31 (1.52)	+0.44 (0.65)	+0.87 (1.46)	+0.48 (0.61)
Missing	44				
Stove type					
Electric	3,698	+0.29 (0.18)	−0.02 (0.07)	+0.48 (0.17)	+0.10 (0.07)
Gas	3,360	−0.27 (0.19)	+0.06 (0.07)	−0.65 (0.18)	+0.19 (0.07)
Both	563	−0.12 (0.45)	+0.08 (0.17)	−0.48 (0.44)	+0.26 (0.17)
Missing	213				

TABLE 4

Regression coefficients (in per cent, with standard errors) for four different measures of parental cigarette smoking and for gas stove exposure in explanatory models for level and growth rate of ln(FEV_1) and ln(FVC)

Variable	FEV_1		FVC	
	Level	Growth rate	Level	Growth rate
*Measures of exposure to parental smoking**				
Model 1				
Maternal smoking status	−0.61 (0.27)	−0.09 (0.11)	0.46 (0.27)	−0.14 (0.11)
Paternal smoking status	−0.41 (0.28)	−0.02 (0.11)	−0.12 (0.27)	0.09 (0.11)
Model 2				
Maternal smoking status	−0.80 (0.25)	−0.10 (0.10)	0.34 (0.25)	−0.13 (0.10)
Model 3				
No. of smokers	−0.47 (0.16)	−0.09 (0.07)	0.16 (0.16)	−0.02 (0.07)
Model 4				
Maternal smoking level (packs/day)	−0.81 (0.21)	−0.17 (0.09)	0.33 (0.21)	−0.17 (0.08)
Exposure to gas stove†				
Presence of a gas stove	−0.41 (0.32)	0.09 (0.13)	−0.56 (0.31)	0.06 (0.12)

* All models include city and parental education.

† All models include city, parental education, and maternal smoking status.

sion analysis including maternal smoking level, city of residence, and socioeconomic status gave an estimated effect on FEV_1 level of −0.81 per cent ($p = 0.0001$) and on growth rate of −0.17 per cent per year ($p = 0.05$) per pack of cigarettes (table 4). These results are qualitatively consistent with those achieved using parental smoking status but more highly significant, as would be expected when substituting a measured for a discrete exposure variable. The maternal smoking level is a measure of total cigarettes smoked per day by the child's mother, and may overestimate smoking in the home. The reversal seen at 46+ cigarettes per day (table 3) may be due to the small sample size in this group and overestimation of the child's exposure. When considered alone, paternal smoking level was not a significant predictor of level or growth rate.

No statistically significant associations were found between FVC values and parental smoking status (table 4). The estimated effect of maternal smoking level on FVC level was +0.33 per cent per pack ($p = 0.12$) and the estimated effect on growth rate was −0.17 per cent per year per pack ($p = 0.04$).

Gas cooking

Mean values of the pulmonary function measures were calculated for children exposed to only one stove type and for children exposed to both electric and gas stoves (table 3). Weighted regression analysis controlling for amount of maternal smoking as well as for city and parental education found no significant associations between gas stove exposure and pulmonary function measures (table 4). The estimated effects of gas stove exposure on pulmonary function level were −0.41 per cent ($p = 0.41$) for FEV_1 and −0.56 per cent ($p = 0.07$) for FVC. Stove type was not significantly associated with pulmonary function growth rates.

DISCUSSION

Although numerous cross-sectional studies have found an association between childhood respiratory health and parental smoking habits, those studies have been unable to assess the degree to which pulmonary function growth rates may be affected by exposure to sidestream cigarette smoke. Tager et al. (16) in a longitudinal study reported that children exposed to one

pack per day of maternal smoking experienced significant deficits in FEV_1 growth rate which resulted in an estimated cumulative deficit in FEV_1 level of 173 ml by adult life relative to children of nonsmoking mothers. This represents a deficit of 3 to 4 per cent in maximum attained level of FEV_1 in early adult life.

Our data indicate that children of mothers smoking one pack per day have an estimated 0.81 per cent lower FEV_1 level at eight years of age than children of nonsmoking mothers ($p = 0.0001$). Furthermore, the estimated effect on FEV_1 growth rate is −0.17 per cent per pack per year of exposure between ages six and 10 ($p = 0.05$). This corresponds to a deficit in FEV_1 growth rate of about 3 ml between the eighth and ninth birthdays for a boy with an initial FEV_1 of 1.62 liters. It also implies a 1.14 per cent lower FEV_1 by age 10, or about 21 ml for a child whose predicted FEV_1 level in the absence of smoking exposure is 1.90 liters. Extrapolation of this effect to age 20 would imply a cumulative effect of 2.8 per cent. Although these results are consistent in direction with the findings of Tager et al. (16) in their study based in East Boston, MA, the estimated effect of maternal smoking on FEV_1 growth rate is only marginally significant in our data, and the size of that effect during the preadolescent years is substantially smaller than reported by Tager et al. This difference may be explained in part by a more substantial effect of parental smoking during adolescence, which cannot be observed in our preadolescent data. The use by Tager and colleagues of a nonlinear model which implies that the largest effect occurs in the early years of exposure is also a factor in the contrasting results for preadolescents. Our model assumes that the incremental effect of exposure to a certain level of cigarette smoking, expressed in percentage terms, is independent of age and exposure history. The relatively brief follow-up interval of four years analyzed for this report does not allow discrimination between these linear and nonlinear exposure-response models. Although the estimated associations between paternal smoking and FEV_1 measurements were similar in direction to those seen for maternal smoking, they were consistently smaller and failed to achieve statistical significance.

The observed association of both FEV_1 level and rate of growth with current maternal smoking behavior could be explained either as a consequence of prenatal and early childhood exposure, which shifted children toward a lower track of pulmonary function growth, or as an effect of exposure during the period of follow-up. Presumably, most women who smoked during the period of follow-up had also smoked when their children were quite young, although some may have abstained during pregnancy. Indeed, only 400 of 7,834 mothers reported a change in smoking status between the first and last examination, an interval of two to four years.

To distinguish between the effects of current and early childhood exposure to cigarette smoke would require follow-up, preferably beginning during pregnancy or in early childhood, of a group of children whose exposure varied substantially over the period of follow-up. In the extreme, if mothers remained either smokers at a fixed smoking level or nonsmokers during the entire period from conception to adulthood of their children, current and early childhood exposure would be identical and no such separation of effects would be possible. This is nearly the case in this study, in that few mothers reported a qualitative change in smoking status during the study, and changes in reported smoking level were presumed to be due in large part to variability in reporting. Although several analyses were attempted to assess the effects of changes in smoking level from year to year after controlling for average exposure during the period of follow-up, these analyses showed that the available information was insufficient for this purpose.

The observed associations between FVC

values and parental smoking behavior were somewhat different. A statistically significant positive association between maternal smoking and FVC had been seen in earlier cross-sectional analyses of data from the first two examinations (7), but the longitudinal data indicate that this positive association was smaller and no longer statistically significant by eight years of age due to a significant negative association between maternal smoking level and FVC growth rate. These results suggest that, if parental smoking leads to increased FVC levels in children up to age six, these effects may disappear as children mature. This pattern contrasts with the apparent increase in the cumulative effect of passive smoking on FEV_1 as children mature.

Other cross-sectional studies have reported increased FVC levels in children exposed to cigarette smoke (25). In a recent report of physiologic measurement in infants, children of smoking mothers had larger functional residual capacities than comparable infants of nonsmoking mothers (26). Lung size has also been found to be increased in offspring of rats who received nicotine throughout pregnancy (27). The relevance of these findings to the observed association between FVC level and passive smoking is uncertain, however, and the association may be a chance occurrence.

Our data suggest that exposure to gas stoves has comparable effects on FEV_1 and FVC. Previous analyses had detected significantly lower levels of both pulmonary function indices at the first examination among children living in homes with gas stoves (7). The longitudinal data, however, show a small and nonsignificant positive association between gas stove use and pulmonary function growth rates, so that by the age of eight years the association between gas stove exposure and FVC level, while still negative, was not statistically significant. These data could be interpreted as suggesting an effect of gas stove exposure among the youngest children which is partially reversible. For the present, however, this interpretation can be only tentative. The limitations of classification by stove type as a surrogate for measured exposure to nitrogen dioxide or other emission products are now widely recognized. Studies using personal monitoring have shown that less than 5 per cent of the interindividual variation in levels of measured NO_2 is explained by stove type, while up to 60 per cent can be explained by measurements in the home (28). Thus, future studies of NO_2 exposure should incorporate indoor or personal monitoring.

The results reported here are based upon a large sample of preadolescent children followed for a relatively short period of time. Nevertheless, they suggest that the effects on FEV_1 of exposure to parental cigarette smoking may be cumulative during the preadolescent years. Moreover, children whose pulmonary function level is reduced due to early environmental exposures may show normal growth in percentage terms but a growing deficit in absolute terms (liters) relative to the levels that would have been achieved in the absence of early effects. Thus, these data are consistent with the hypothesis that children with deficient lung function levels in early childhood fail to reach their full pulmonary function potential. One prevalent hypothesis is that adults who are at risk of developing obstructive airways disease upon exposure to risk factors such as personal cigarette smoking come in large part from the pool of persons affected by childhood exposures. Indeed, data obtained retrospectively in adult samples suggest that childhood events are associated with adult disease (29, 30).

Insight into the validity of this hypothesis will come from continued follow-up and evaluation of these children, particularly as they pass through adolescence. Personal cigarette smoking will, unfortunately, become a factor for 20 per cent of these children (31). The data collected from these children will provide an opportunity to assess the association between early child-

hood exposures and the response to personal cigarette smoking.

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